26 Ploem, J.S., Reflection-contrast microscopy as a tool for investigation of the attachment of living cells to a glass surface, in: Mononuclear phagocytes in immunity, infection and pathology, pp.405–421. Ed. R. van Furth. Blackwell Scientific Publications, Melbourne and London 1975.
27 Morgan, M.J., and Faik, P., Carbohydrate metabolism in cultured animal cells. Biosci. Reps *1* (1981) 669–686.
28 Mc Keehan, W.L., Glycolysis, glutaminolysis and cell proliferation. Cell Biol. int. Reps *6* (1982) 635–650.
29 Tairbekov, M.G., and Parfyonov, G.P., Cellular aspects of gravitational biology. Physiologist *24* (1981) S69–S72.
30 Tixador, R., Richoilley, G., Grechko, G., Nefedov, Y., and Planel, H., Multiplication de *Paramecium aurelia* a bord du vaisseau spatial Saliout-6 (Expérience Cytos). C.r. Acad. Sci. Paris *D287* (1978) 829–832.
31 Wolosewick, J.J., and Porter, K.R., Microtrabecular lattice of the cytoplasmic ground substance. Artifact or reality. J. Cell Biol. *82* (1979) 114–139.
32 Pollard, E.C., Physical determinants of receptor mechanism, in: Gravity and the organism, pp.25–34. Eds A. Gordon and M.J. Cohen. University of Chicago Press, Chicago 1971.
33 Wohlfarth-Bottermann, K.E., Differentiations of the ground cytoplasm and their significance for the generation of the motive force of amoeboid movement, in: Primitive motile systems in cell biology, pp.79–109. Eds R.D. Allen and N. Kamiya. Academic Press, New York and London 1964.
34 Schatz, A., and Teuchert, G., Effects of combined O-g simulation and hypergravity on eggs of the nematode *Ascaris suum*. Aerospace Medicine *43* (1972) 614–619.

0014-4754/83/121323-07$1.50+0.20/0

Control of corpus allatum activity in *Diploptera punctata:* roles of the pars intercerebralis and pars lateralis

R.P. Rüegg, D.J. Lococo and S.S. Tobe

Department of Zoology, University of Toronto, 25 Harbord Street, Toronto (Ontario, Canada M5S 1A1), May 17, 1983

Summary. Control of the corpora allata (CA) of *Diploptera punctata* is maintained by at least 2 factors. The glands are directly inhibited by an allatostatin arriving at the CA via the nervi corporis cardiaci I (NCC I). Destruction of the putative source (median neurosecretory cells, MNC) of the allatostatin by radio-frequency (RF) cautery relieved the inhibition imposed on the CA of virgin females, and the glands became active. Similarly, destruction of the lateral neurosecretory cells (LNC) also relieved the inhibition. We propose that the LNC stimulated the MNC to release allatostatin. RF-cautery did not result in the activation of CA of pregnant or ovariectomized females. Activation of the CA may therefore require not only absence of the inhibitory factor but also the presence of a stimulatory one (perhaps from the ovary).

Introduction

In the adult female cockroach *Diploptera punctata*, the stimulus of mating enhances the biosynthesis of juvenile hormone (JH) by the corpora allata (CA) and results in a cycle of hormone production and subsequent oocyte growth. JH biosynthesis may be controlled by inhibitory substances (allatostatins), stimulatory substance(s) (allatotropins) or both. In most species studied, nervous connexions (i.e. nervi corporis cardiaci I, NCC I: nervi corporis cardiaci II, NCC II) from the brain to the CA appear to control CA activity. For example, nervous connexions appear to be stimulatory in *Schistocerca paranensis*[28,29], *S. gregaria*[35] and *Locusta migratoria*[11]. On the other hand, centers in the brain and their axonal extensions to the CA appear to be inhibitory in *Leucophaea maderae*[3], *Diploptera punctata*[4,25], *Gryllus domesticus*[13] and *Nauphoeta cinerea*[15,16] to name but a few examples.

Destruction of a stimulatory cerebral center can provide for an overall decrease in relative CA activity whereas destruction of an inhibitory center can result in an overall increase in activity. For example, cautery of the lateral neurosecretory cells (LNC) or severance of the NCC II prevents normal oocyte growth in *S. paranensis*[28,29]. Destruction of the axons from the medial neurosecretory cells (MNC) prevents the normal cycle of JH biosynthesis in *L. migratoria*[11]. Conversely, destruction of the axons from the MNC stimulates oocyte development in *L. maderae*[4].

Following denervation, the CA of *D. punctata* become more biosynthetically active and a normal cycle of JH biosynthesis ensues, resulting in oocyte development and oviposition[25,36]. Thus, innervation of the gland is not necessary for either the increase in JH biosynthesis at the beginning of the cycle or the decrease at the end. In addition to the inhibitory influence of the NCC I, it has also been demonstrated that the ovaries have a stimulatory effect on the CA and are necessary for a normal gonotrophic cycle[26,27]. Recent evidence indicates that the stimulatory effect of the ovary acts directly on the CA and not via the brain[21,22].

It is presently believed that JH biosynthesis by the CA of *D. punctata* may be controlled by direct neurohormonal inhibition or by neurohormonal or neural

blocks on release of stimulatory neurohormones[31]. Similarly, the decrease in JH biosynthesis observed at the end of a gonotrophic cycle may be effected by either an inhibitory factor or the absence of a stimulatory one[34]. It was the goal of the present paper to examine these hypotheses and to investigate the cerebral control of juvenile hormone biosynthesis. To this end, we have determined the effect of radio-frequency (RF) cautery of selected cerebral centres on JH biosynthesis by the CA of *D. punctata*. We have demonstrated that selective destruction of 1. the pars intercerebralis (PI) or 2. axons of the MNC, 3. the pars lateralis (PL) or 4. the axonal projections from the PL to the PI relieved the inhibition normally occurring on the CA of virgin females. Subsequently, the CA underwent a cycle of JH biosynthesis characteristic of mated females. Stimulation of JH biosynthesis by cautery of cerebral centres was not effective in ovariectomized or pregnant females. These data suggest that in addition to inhibitory cerebral control of the CA, other factors are involved in the feedback control of JH biosynthesis.

Materials and methods

The colony of *D. punctata* was maintained as previously described[30]. Female last instars were isolated from males prior to adult emergence in order to obtain virgin female adults. Ovariectomy was performed on last larval instars as previously described[26].

Radio-frequency (RF) cautery. Electrocautery was performed using a Gebrüder-Martin Electrotrom 60. Virgin females and ovariectomized, mated females were cauterized 2 days after the imaginal molt. Pregnant females were cauterized 10 days after ovulation. Females were anesthetized on ice and mounted on an operating block covered with aluminum foil. To provide good conductivity, the insect was restrained with a piece of foil and the entire mounting grounded. A small rectangular window was cut in the frons of the insect and the tracheae and fat body overlying the brain removed. All hemolymph on the surface of the brain was removed using an aspirator pulled from a Pasteur pipette. Once free of hemolymph, the radio-frequency electrode made from fine tungsten wire (50 µm) was placed on the area to be cauterized and a current applied for 250 msec. A few crystals of streptomycin were placed in the wound, and the cuticle overlying the brain replaced and the insect allowed to recover. Sham operations were performed using the same procedure except that no current was applied to the electrode.

Radiochemical assay. At various times after electrocautery, biosynthesis of juvenile hormone by the isolated CA was measured using an in vitro radiochemical assay based on the incorporation of the methyl moiety of [methyl-^{14}C]-methionine (Amersham Searle; sp. act. 56.7 mCi/mmole; final sp. act. $\simeq$ 38 mCi/mmole) into C_{16}JH (JH III) after a 3-h incubation period[20,32]. JH was extracted from the incubation medium using the partition assay of Feyereisen and Tobe[6]. At the time of assay, the lengths of the basal oocytes were measured as a bioassay for the cumulative effect of JH titres[33].

Histology. Following removal of the CA for radiochemical determination of biosynthetic activity, the heads of individual insects were fixed overnight in Bouins TCA. The brains were then embedded in paraffin and sectioned at 5 µm. The sections were stained with paraldehyde fuchsin and victoria blue[10], mounted and examined for neurosecretory material and lesion size and location.

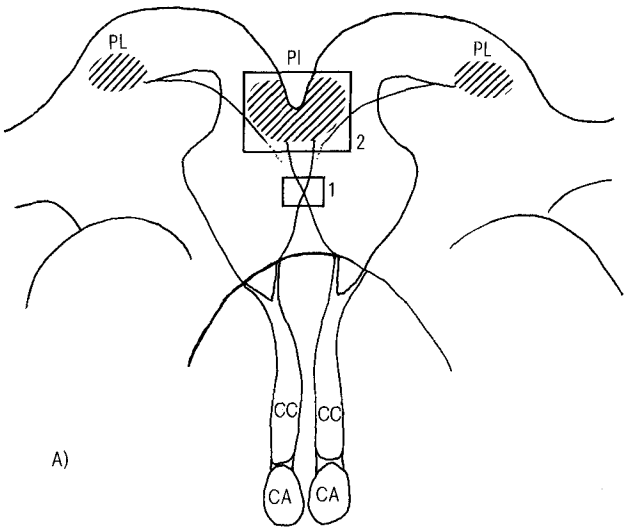
Nickel chloride backfilling. In some insects selected cerebral centers were cauterized unilaterally; axons from the CA-CC complex were backfilled with nickel chloride[17] 4 or 5 days after the operation. These preparations were examined to determine the relative location of the cautery lesion; this information was related to basal oocyte length of the respective animal. In other insects, axons were backfilled from unilaterally sectioned nervi corporis allati I (NCA I) and examined in wholemount to determine the extent of axonal and/or dendritic communication between neurons in the PI and PL.

Results

a) RF-cautery of the MNC axons. Following RF-cautery of the MNC tracts at the point of decussation (see map, fig. 1A) the CA of virgin *D. punctata* underwent an apparently normal cycle of JH biosynthesis which was accompanied by a corresponding increase in oocyte length (fig. 2). Within 24 h of the destruction of the MNC axons, the CA synthesized JH at a rate significantly (Students t-test, $p < 0.05$) greater than sham-operated females. As in normal mated females, the rate of JH biosynthesis peaked 4–5 days after the operation and then declined to unstimulated rates (i.e. prior to operation) by day 7 or 8.

b) RF-cautery of the PL and axonal projections. Attempts to unilaterally cauterize the PI invariably resulted in activation of the CA on the ipsilateral side (fig. 1B). Since the axons of the MNC decussate within the brain, this was a surprising result. However, examination of operated brains which had been subjected to backfilling with nickel chloride[17] showed that the PI had not been cauterized but rather, an area adjacent to it. This suggested that perhaps another input into the PI was controlling the CA. Since some axons from the PL lie in close proximity to the axons of the MNC (fig. 5A), the effect of cautery on the PL and associated axons was examined. As in the destruction of the MNC axons, destruction of the PL axons (fig. 1B) stimulated the biosynthetic activity of the CA.

Another series of experiments was performed in which the PL was cauterized unilaterally rather than



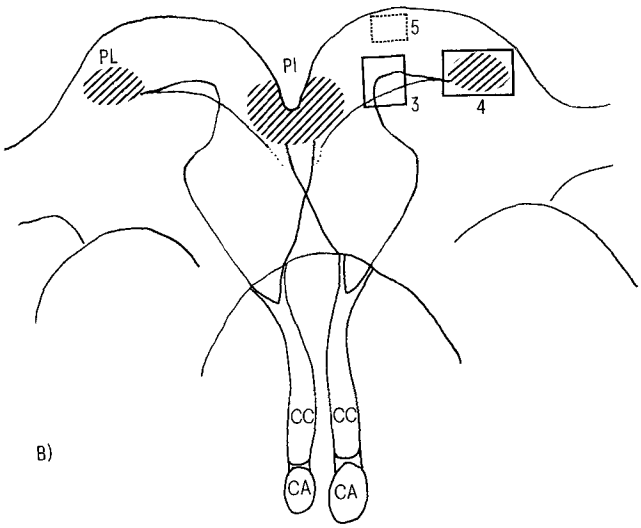


Figure 1. *A* Diagram showing location and effect of PI cautery. RF cautery of either the MNC axons at the point of decussation (1) or the MNC bodies (2) results in the hypertrophy and increased activity of both corpora allata.

B Diagram showing location and effect of unilateral LNC cautery. RF cautery of the PL axons (3) or PL (4) results in the hypertrophy and increased activity of the ipsilateral gland only. Control cautery (5) had no effect. PL, pars lateralis; PI, pars intercerebralis; CA, corpus allatum; CC, corpus cardiacum.

the axons (fig. 1B). In these experiments, the contralateral CA was used as a control and each CA was incubated individually. As can be seen in figure 3, the ipsilateral CA underwent a normal cycle of JH biosynthesis, whereas the contralateral gland synthesized JH at low levels throughout the experimental period.

c) Effect of PL cautery on pregnant or ovariectomized virgin females. Within 4 days of cautery of the PL, the CA of normal or sham ovariectomized virgin females synthesized JH at rates of 40–50 pmoles h^{-1} (fig. 4). However, the same operation on either ovariectomized or pregnant females (18 days after mating, 10 days after ovulation) did not result in an elevation in hormone biosynthesis above the unstimulated level of about 10–15 pmoles h^{-1}.

d) Histology. Extensive disruption of the brain structure made it difficult to identify any one cell type in sections of brains of cauterized animals. The lesions caused by cautery were generally 100–150 µm in width and 50–75 µm in depth and filled with hemocytes. Whether operated or control-operated, the disruption seen within the brain was always restricted to the hemisphere which was cauterized. Since similar disruptions were observed in control-cauterized females, in which the CA did not become active, the cautery did not appear to interfere with the normal inhibition of the CA.

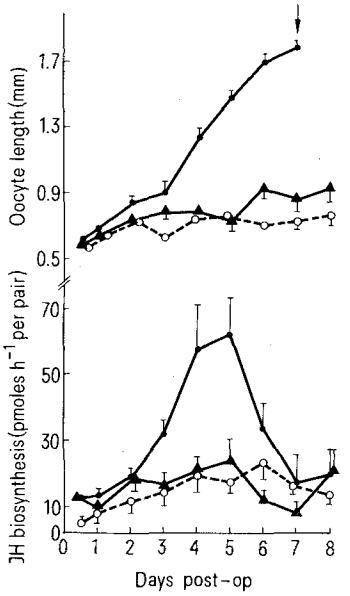


Figure 2. Effects of cautery of the MNC axons on oocyte length and rates of juvenile hormone biosynthesis. Each point represents the mean±SEM for at least 8 determinations for unoperated (○), control-operated (▲) and operated (●) virgin females. The arrow indicates the time of oviposition for the operated group.

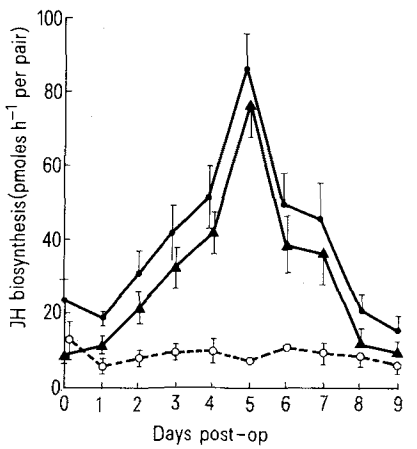


Figure 3. Mean rates of juvenile hormone biosynthesis for the ipsilateral gland (▲), the contralateral gland (○) and their sum (●) following unilateral PL cautery. Each point represents the mean±SEM for at least 5 determinations.

e) Backfilling. Examination of many preparations which were backfilled has provided clear evidence for a role by the PL in the control of CA activity. Unilateral cautery of the PL or axons always resulted in significant oocyte growth in the operated females (figs 5B, 5C). In cases where some axons of the PL were destroyed (fig. 5B), backfilling occurred up to the point of cautery but not beyond (to the cell bodies). However, following cautery of an area adjacent to the axons (fig. 5C), the cell bodies of the PL filled and no oocyte growth was observed.

Because the PI appears to regulate CA activity, and because cautery of the PL also stimulated CA activity, we hypothesized that some synaptic communication exists between the 2 groups of cells. Figure 5A shows a wholemount of a brain which was unilaterally backfilled with nickel chloride from a sectioned NCA I. In every case, cells in the contralateral PI and ipsilateral PL backfilled with nickel. Neuronal projections from the PL can also be seen to extend ventromedially towards the ipsilateral PI and to the axon tracts from the cells of the contralateral PI.

Discussion and conclusions

It is clear from the results of this paper that the CA of adult *D. punctata* are controlled by at least 2 substances: an inhibitory substance produced in the PI, and a stimulatory substance which occurs only in the presence of the ovaries. These results are consistent with the evidence which shows that intact nerves are necessary to prevent activation of the CA in virgin female *D. punctata*[24]. It also appears probable that the release of an inhibitory factor from the MNC (allatostatin) is controlled by a stimulatory influence from the PL. It is therefore likely that the PL receives

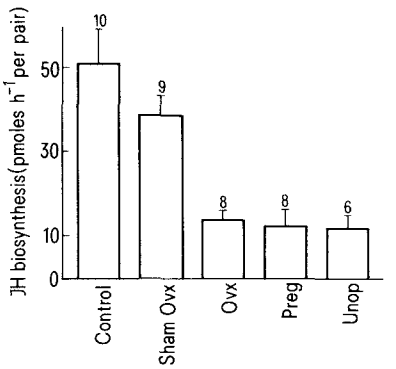


Figure 4. Mean rates of juvenile hormone biosynthesis for PL-cauterized females: virgins (control), sham ovariectomized virgins (Sham Ovx), ovariectomized virgins (Ovx), pregnant females (Preg). Also shown are unoperated virgin females (Unop). Determinations were made 4 days after the operation. Sample size is indicated above the narrow vertical bars, which represent SEM.

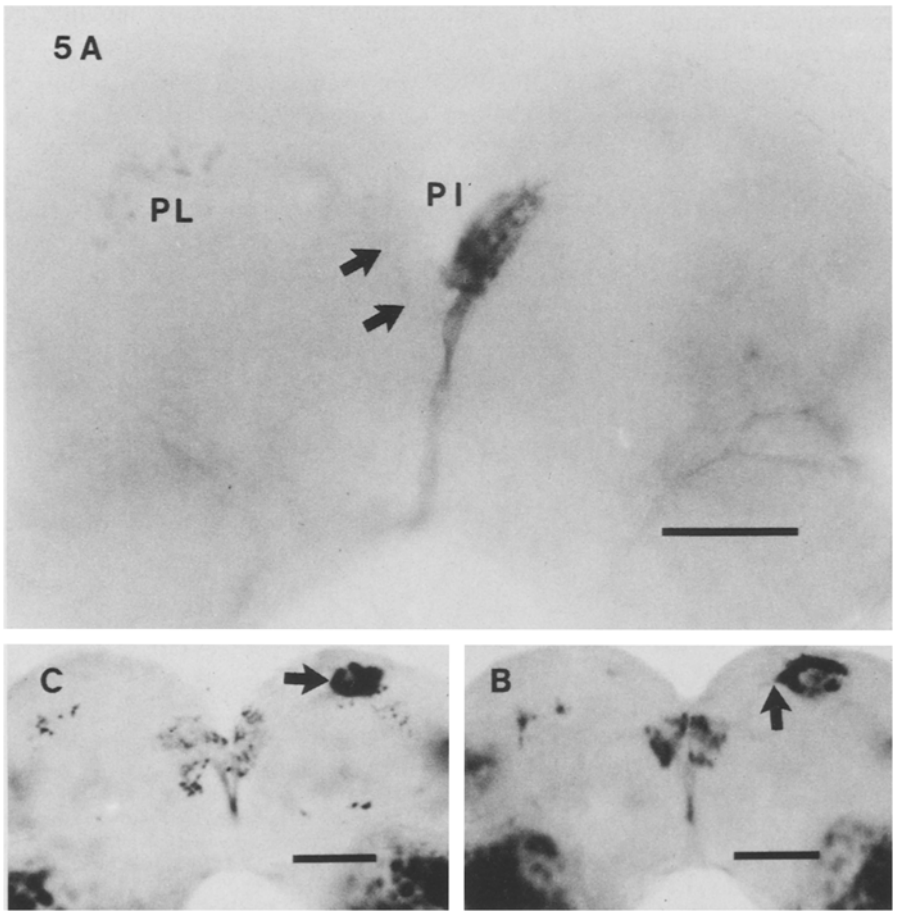


Figure 5. *A* Wholemount of a brain backfilled unilaterally with nickel chloride. Note that only the PL on the ipsilateral side and the MNC on the contralateral side backfilled. Also note that axons from the PL followed two routes – downward (posteriorly) to the NCC II and ventromedially (arrow) towards the MNC.

B Representative wholemount of a unilaterally cauterized brain backfilled bilaterally with nickel chloride; cautery has destroyed the PL axons. Note that backfilling occurred only to the lesion (arrows) and not beyond. Within 5 days of cautery, the basal oocytes of this insect had grown to 1.68 mm.

C Representative wholemount of a unilaterally cauterized brain backfilled bilaterally with nickel chloride; the cautery lesion (arrow) has barely missed the PL axons. Note that the PL filled bilaterally. Within 5 days of cautery, the basal oocytes of this insect had not grown beyond 0.67 mm (i.e. non-vitellogenic). PI, pars intercerebralis; PL, pars lateralis. Scale bars represent 250 µm.

neural information from the stimulus of mating which, in normal mated females causes the cessation of stimulatory signals from the PL. In the absence of this stimulation (as a result of mating), the MNC are no longer stimulated to produce/release the allatostatin and the CA become active. This, too, is consistent with the view that denervation of the CA mimics mating in *D. punctata*[4,22].

In the absence of inhibitory signals, activation of the CA still requires the presence of a stimulatory factor, because in ovariectomized females, neither denervation of the CA[27] nor cautery of the cerebral inhibitory centres (this paper) alone results in the activation of the CA. This stimulatory factor appears to be present only in females in which the ovaries are maturing[21,22,27,31]. This hypothesis is similar to that suggested by Lanzrein and her colleagues in *N. cinerea*[15,16].

The mechanism which inhibits the CA early in the gonotrophic cycle or its equivalent is clearly different from the mechanism which is responsible for the decline in, and suppression of activity at the end of the gonotrophic cycle. For example, neither denervation of CA[25] nor cautery of inhibitory cerebral centres (this paper) resulted in activation of the CA of pregnant females. However, denervated CA of pregnant females become active when implanted into day-0-mated females[25]. We do not know whether a 2nd inhibitory substance, the lack of a stimulatory factor, or both are responsible for the decline of CA biosynthetic activity and the suppression of the CA throughout pregnancy. In any event, the inhibitory influence takes precedence over the stimulatory one because unilateral cautery resulted in the activation of only the ipsilateral CA (fig. 3). It is interesting to note that in PL-cauterized virgin females, abortion eventually occurred and this event was followed by a 2nd, apparently normal, gonotrophic cycle (Rüegg, unpublished).

How might the PL regulate MNC? Based on electrophysiological studies, Bruce and Wilkens[2] have suggested that optic and other sensory areas provide input into the MNC of *Sarcophaga bullata*. Some of the most convincing evidence, however, is provided by unilateral backfills of the NCC I and II of *Schistocerca vaga* and *Periplaneta americana*[14]. These authors observed that in *S. vaga*, collaterals of the MNC project towards the LNC and that fibers from the LNC can be described as 'terminating among the NCC I fibers lateral to the NCC I'. The close proximity of fibers from NCC I and II in the dorsal protocerebral neuropile and along NCC I suggests that either both receive similar input or that they interact. In *P. americana* it appears that fibers from NCC I overlap fibers of NCC II[4,8,14,19].

The arrangement of axons projecting from the PL and PI in *D. punctata* is similar to that of *P. americana* and *S. vaga*. In figure 5A fibers from the PL can be seen to overlap fibers of the MNC from the contralateral side. Our evidence suggests that the PL provides input into the contralateral MNC as opposed to directly inhibiting the CA via the axons of the NCC II, because the NCC I but not NCC II appears to regulate the CA in *D. punctata*[36].

Although activation of the CA in *S. paranensis*[28,29], *S. gregaria*[35], *L. migratoria*[10,18] and *Pyrrhocoris apterus*[12] require intact NCA, the CA of *L. maderae*[5] and *D. punctata*[23,36] do not. In the latter cases, only inhibition alone requires intact NCA I or NCC I. On the other hand, it appears that the stimulatory influence of the ovaries occurs directly on the CA of *D. punctata* because those CA which are denervated[27] or have nervous input destroyed by cautery are still able to respond to the stimulatory stimulus. Similarly, Lanzrein et al. have demonstrated that in decapitated females of *N. cinerea*, implanted CA become active only in the presence of an ovary[15]. Also, Hodkova[12] has suggested that inactivation of the CA of *Pyrrhocoris apterus* is due to the absence of stimulation and direct nervous inhibition.

In conclusion, it seems probable that the CA of *D. punctata* are primarily controlled by an inhibitory substance (allatostatin) which is released from the MNC. This inhibitory control may be similar to that suggested by Scharrer[24] – a neurosecretory signal employed for long-term inhibition of an endocrine gland. The PL apparently provides a stimulatory input to the MNC which sustains the inhibitory flow to the CA. In the absence of inhibition (due to mating, denervation of the CA, or cautery of cerebral control centres) activation of the CA also requires a stimulatory factor from the ovary. It is clear that the decline in CA activity at the end of the gonotrophic cycle does not require nervous connexions from the brain. Whether this decline is due to the absence of a stimulatory factor, the presence of a humoral inhibitory factor, or both[31], remains to be determined.

Acknowledgments. We wish to thank Prof. Barbara Stay for performing the ovariectomies and for many useful comments and helpful discussion. We also wish to thank Professor Werner Loher for critically reading the manuscript. This work was supported by the Natural Sciences and Engineering Research Council of Canada.

1 Baehr, J.C., Contrôle neuroendocrine du fonctionnement du corpus allatum chez *Rhodnius prolixus*. J. Insect Physiol. *19* (1973) 1041–1055.
2 Bruce, I.C., and Wilkens, J.L., Neuronal activity in neurosecretory cells of the fleshfly, *Sarcophaga bullata*. J. comp. Physiol. *112* (1976) 109–122.
3 Englemann, F., Die Steuerung der Ovarfunktion bei der ovoviviparen Schabe *Leucophaea maderae* (Fabr.). J. Insect Physiol. *1* (1957) 257–278.
4 Englemann, F., The control of reproduction in *Diploptera punctata* (Blattaria). Biol. Bull. mar. biol. Lab. Woods Hole *116* (1959) 406–419.
5 Englemann, F., and Lüscher, M., Die hemmende Wirkung des Gehirns auf die Corpora allata bei *Leucophaea maderae* (Orthoptera). Verh. dt. zool. Ges. (1956) 215–220.

6 Feyereisen, R., and Tobe, S.S., A rapid partition assay for routine analysis of juvenile hormone release by insect corpora allata. Analyt. Biochem. *111* (1981) 373–375.

7 Fraser, J., and Pipa, R., Corpus allatum regulation during the metamorphosis of *Periplaneta americana:* Axon pathways. J. Insect Physiol. *23* (1977) 975–984.

8 Fraser, J., and Pipa, R., Location and central projections of neurons associated with the retrocerebral neuroendocrine complex of the cockroach *Periplaneta americana* (L). Cell Tiss. Res. *193* (1978) 443–445.

9 Ganagarajah, M., and Saleuddin, A.S.M., A simple manoeuver to prevent loss of sections for performic acid - Victoria blue technique and a comparison of various neurosecretory stains. Can. J. Zool. *48* (1970) 1457–1458.

10 Girardie, J., and Girardie, A., Neurosécrétion médiane après section des nerfs hypocérébrocardiaques chez le criquet migrateur. C.r. Acad. Sci. Paris *284* (1977) 1433–1436.

11 Girardie, J., Tobe, S.S., and Girardie, A., Biosynthèse de l'hormone juvenile et maturation ovarienne chez le criquet migrateur. C.r. hebd. Seanc. Acad. Sci. Paris *293* (1981) 443–446.

12 Hodkova, M., Nervous inhibition of corpora allata by photoperiod in *Pyrrhocoris apterus.* Nature *263* (1976) 521–523.

13 Huignard, J., Recherches histophysiologiques sur le contrôle hormonal de l'ovogenèse chez *Gryllus domesticus* (L.). C.r. Acad. Sci. *259* (1964) 1557–1560.

14 Koontz, M., and Edwards, J.S., The projection of neuroendocrine fibers (NCC I and II) in the brains of three Orthopteroid insects. J. Morphol. *165* (1980) 285–299.

15 Lanzrein, B., Wilhelm, R., and Buschor, J., On the regulation of the corpora allata activity in adult females of the ovoviviparous cockroach, *Nauphoeta cinerea;* in: Juvenile Hormone Biochemistry, pp. 147–160. Eds G. Pratt and G. Brooks. Elsevier North-Holland Press, Amsterdam 1981.

16 Lanzrein, B., Wilhelm, R., and Gentinetta, V., Int. Conference on the Regulation of Insect Development and Behaviour, Part II, pp. 523–534. Wroclaw Technical Univ. Press, Wroclaw, Poland 1980.

17 Lococo, D.J., and Tobe, S.S., Neuroanatomy of the Brain/Retrocerebral complex, in Particular the pars intercerebralis and partes lateralis in the cockroach, *Diploptera punctata* (Dictyoptera: Blaberidae). Int. J. Insect Morphol. Embryol. (1983), in press.

18 Moulins, M., Girardie, A., and Girardie, J., Manipulations of sexual physiology by brain stimulation in insects. Nature *250* (1974) 339–340.

19 Pipa, R.L., Locations and central projections of neurons associated with the retrocerebral neuroendocrine complex of the cockroach *Periplaneta americana* (L.). Cell Tissue Res. *193* (1978) 443–455.

20 Pratt, G.E., and Tobe, S.S., Juvenile hormone radiobiosynthesized by corpora allata of adult female locusts *in vitro.* Life Sci. *14* (1974) 575–586.

21 Rankin, S., and Stay, B., Effects of decapitation and ovariectomy on the regulation of juvenile hormone synthesis in the cockroach *Diploptera punctata.* J. Insect Physiol. (1983) in press.

22 Rankin, S.M., and Stay, B., The changing effect of the ovary on rates of juvenile hormone synthesis in *Diploptera punctata.* Gen. comp. Endocr. (1983) in press.

23 Roth, L.M., and Stay, B., Oocyte development in *Diploptera punctata* (Eschscholtz) (Blattaria). J. Insect Physiol. *7* (1961) 186–202.

24 Scharrer, B., Neural control of endocrine glands in invertebrates. Proc. IVth int. Congr. Endocr. *273* (1973) 210–214.

25 Stay, B., and Tobe, S.S., Control of juvenile hormone biosynthesis during the reproductive cycle of a viviparous cockroach. I. Activation and inhibition. Gen. comp. Endocr. *33* (1977) 531–540.

26 Stay, B., and Tobe, S.S., Control of juvenile hormone biosynthesis during the reproductive cycle of a viviparous cockroach. II. Effects of unilateral allatectomy, implantation of supernumerary corpora allata, and ovariectomy. Gen. comp. Endocr. *34* (1978) 276–286.

27 Stay, B., Tobe, S.S., Mundall, E.C., and Rankin, S., Ovarian stimulation of juvenile hormone biosynthesis in the viviparous cockroach, *Diploptera punctata.* Gen. comp. Endocr. (1983) in press.

28 Strong, L., The relationships between the brain, corpora allata, and the oocyte growth in the Central American locust, *Schistocerca* sp. I. The cerebral neurosecretory system, the corpora allata, and oocyte growth. J. Insect Physiol. *11* (1965) 135–146.

29 Strong, L., The relationship between the brain, corpora allata, and oocyte growth in the Central American locust, *Schistocerca* sp. II. The innervation of the corpora allata, the lateral neurosecretory complex, and oocyte growth. J. Insect Physiol. *11* (1965) 271–280.

30 Szibbo, C.M., and Tobe, S.S., The mechanism of compensation in juvenile hormone synthesis following unilateral allatectomy in *Diploptera punctata.* J. Insect Physiol. *27* (1981) 609–613.

31 Tobe, S.S., Regulation of the corpora allata in adult female insects; in: Insect Biology in the Future, pp. 345–367. Academic Press, New York 1980.

32 Tobe, S.S., and Pratt, G.E., The influence of substrate concentrations on the rate of insect juvenile hormone biosynthesis by corpora allata of the desert locust *in vitro.* Biochem. J. *144* (1974) 107–113.

33 Tobe, S.S., and Stay, B., Corpus allatum activity in vitro during the reproductive cycle of the viviparous cockroach, *Diploptera punctata* (Eschscholtz). Gen. comp. Endocr. *31* (1977) 138–146.

34 Tobe, S.S., and Stay, B., Control of juvenile hormone biosynthesis during the reproductive cycle of a viviparous cockroach. III. Effects of denervation and age on compensation with unilateral allatectomy and supernumerary corpora allata. Gen. comp. Endocr. *40* (1980) 89–98.

35 Tobe, S.S., Chapman, C.S., and Pratt, G.E., Decay in juvenile hormone biosynthesis by insect corpus allatum after nerve transaction. Nature *268* (1977) 728–730.

36 Tobe, S.S., Stay, B., Friedel, T., Feyereisen, R., and Paulson, C., The role of the brain in regulation of the corpora allata in female *Diploptera punctata;* in: Juvenile Hormone Biochemistry, pp. 161–174. Eds G.E. Pratt and G.T. Brooks. Elsevier/North-Holland, Amsterdam 1981.

0014-4754/83/121329-06$1.50 + 0.20/0